Liver steatosis and in-phase/opposed-phase MR imaging: theory and clinical applications at 3T

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Abstract
Liver steatosis may evolve into steatohepatitis then cirrhosis with related complications. It may also contribute to hepatocellular failure, sometimes fatal after major hepatectomy, especially in the setting of liver transplantation with living donor. Imaging must allow non-invasive detection and accurate quantification. In and out of phase MR imaging routinely performed in clinical practice is a simple and robust means of achieving these goals. In this article, we will review the histological, pathophysiological, and clinical features of liver steatosis and the key points of in and out of phase pulse sequences and underlying physical principles. The T2* relaxation, cause of a loss of signal between both echo times must be taken into account. Echo times must be known for image interpretation, and optimized, especially at 3T. Finally, the T1 of lipids and water is different and causes T1 effects that may lead to quantification errors while being advantageous for image interpretation. The combination of these factors allows detection and quantification of liver steatosis in routine clinical practice.

Key words: Liver. MRI. Liver steatosis.
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Subcutaneous fat, for example. This is not a fat overload disorder as is hepatic steatosis but a physiological storage cell, closely resembling a hepatocyte overloaded with lipids. Steatosis can be found in several organs: muscles, heart, kidney, spleen (rarely), but it is more classically reported in the liver (hepatic steatosis).

Two types of steatosis have been described (fig. 1).

Microvesicular (or microvacuolar) steatosis

The cytoplasm of the hepatocytes contains many small vacuoles with a nucleus remaining in the center. This type is most often found in drug etiologies (6, 7) (notably the cyclins, antivirals, valproic acid, and amiodarone), and can be paired with acute hepatocellular failure.

Macrovesicular (or macrovacuolar) steatosis

A voluminous intracytoplasmic lipid vacuole displaces the cell to the periphery. This is the most frequent form of steatosis, mainly caused by viral or autoimmune hepatitis, alcohol, obesity, diabetes, dyslipidemia, or genetic metabolic diseases.

These two types of fatty liver disease illustrate how the disease evolves over time, from microvesicular to macrovesicular. The two are often associated (8).

Metabolism involved in steatosis (fig. 2)

Fatty acids coming from the diet or freed by lipolysis of triglycerides of adipose tissue can be taken up by hepatocytes. They are then oxidized or serve to synthesize different derived molecules (triglycerides, phospholipids, etc.). In parallel, hepatocytes can biosynthesize fatty acids de novo from acetyl CoA via a process called lipogenesis (9).

Hepatocytes can export their excess fatty acids as triglycerides incorporated in lipoproteins (VLDLs) or as ketone bodies (biosynthesized from acetyl CoA coming from oxidation of fatty acids) or as phospholipids secreted in the bile (9, 10).

Hepatic steatosis is the result of an imbalance between the production of triglycerides and their catabolism or their secretion. This imbalance can be explained by:

- Excessive amounts coming from the diet or adipose tissue;
- An increase in lipolysis and input of fatty acids in the hepatocyte;
- An increase in fatty acid synthesis by the hepatocyte (excessive lipogenesis);
- A decrease in synthesis and/or secretion of lipoproteins;
- Insufficient catabolism of fatty acids (β-oxidation and/or insufficient synthesis of phospholipids).

The main causes of increased triglyceride synthesis are diet or parenteral feeding rich in fatty acids, corticosteroids, diabetes, alcoholism, and mitochondrial diseases, although the main causes of reduced triglyceride excretion are toxic sources (alcohol, drugs, toxin), hypoxia (whatever the source), and malnutrition (apoprotein synthesis defect). These etiologies can be associated, although the cause can sometimes be idiopathic.

It is important to remember that steatosis is a reversible process when its cause disappears. On the other hand, chronic steatosis in a steatohepatitis context can evolve toward cirrhosis and its complications (11).

Advantages of quantifying steatosis

Quantifying steatosis is currently of major importance in two cases: nonalcoholic fatty liver disease and live-donor liver transplantation.

Nonalcoholic fatty liver disease (NAFLD) and its consequences

Nonalcoholic fatty liver disease (NAFLD) is the leading cause of chronic liver disease in Western countries, with a prevalence estimated 10% and 24% of the general population (11, 12). Probably occurring after insulin resistance through an increase in adipocyte lipolysis and an increase in hepatocyte captation of fatty free acids (13), nonalcoholic fatty liver disease has recently become one of the elements of insulin resistance syndrome (14). In 20%–30% of cases, NAFLD can be associated with necrotic inflammatory lesions with or without fibrosis, defining nonalcoholic steato-

Fig. 1: Histological specimen of the steatotic liver. Optically empty macrovacuole (asterisk) and optically empty microvacuoles (arrow).

Fig. 2: Simplified diagram of lipid metabolism in the hepatocyte.
hepatitis (NASH). NASH is a potentially serious liver disease that can evolve toward fibrosis in more than 50% of cases and cirrhosis in 15%, with a risk of hepatocarcinoma (11). Quantifying and monitoring steatosis can therefore allow detection and follow-up of NAFLD (1).

**Live-donor liver transplantation**

In Europe, according to Selzner, approximately 20% of patients who should undergo liver resection and 25% of organ donors for transplantation are carriers of liver steatosis (15). Beyond 60% of steatosis, harvesting is contraindicated because of a major risk of hepatocellular insufficiency (16). In cases of less than 30% steatosis, on the contrary, the risk of dysfunction of the transplant is equivalent to that of nonsteatotic livers (16). Between 30% and 60%, the risk is variable and depends on whatever other factors may be associated (15).

Several animal studies (17-19) have investigated therapeutic methods that make it possible to transplant highly steatotic livers (20). In the future, this will lead to pretransplantation evaluation of liver steatosis so as to select patients best able to benefit from these therapies.

**Other potential clinical applications**

Many questions remain. Is hepatic steatosis an additional cardiovascular risk factor (21)? Can a quantitative evaluation of hepatic steatosis be recommended before major liver resection (22)? Can screening and/or follow-up of hepatic steatosis be recommended in patients presenting biological liver anomalies or in those who are on amiodarone, tamoxifen, or antiretrovirals (23)? Do diabetic patients have a therapeutic response profile that differs depending on the rate of fatty liver disease?

**Quantification methods**

The reference method used to evaluate steatosis is histology, an invasive procedure. Several noninvasive techniques have been proposed such as ultrasound, which can detect steatosis with a sensitivity of 83% and specificity of 100% (24) but cannot quantify it; CT, which has a lower sensitivity (54%-85%) (24) and involves irradiation; and finally MRI, with in-phase/opposed-phase MRI, or magnetic resonance spectroscopy (MRS), which is tending to become the standard (4, 5). MRS today appears frequently in clinical studies, but its low availability and its inherent problems mean that IP/OP is considered with much greater attention, and is, in the end, simple to use (contrary to MRS), provides highly reliable results, and is available on all MRI imagers.

**Technical aspects of the in-phase/opposed-phase sequence**

**Principle**

Larmor’s equation (1) provides the resonance frequency (or Larmor frequency) using the following formula:

$$f_0 = \frac{B_0}{\gamma}$$

where $f_0$ is expressed in Hertz, $\gamma$ is the gyromagnetic ratio specific to each nucleus ($\gamma = 2,6751987 \times 10^8$ rad. T$^{-1}$s$^{-1}$ for the proton), and $B_0$ represents the main magnetic field. This frequency represents the frequency with which the water protons precess around $B_0$.

However, depending on the chemical environment in which the protons are placed, the gyromagnetic ratio is slightly modified, which alters the Larmor frequency. For example, the Larmor frequency of lipid protons is 3.25 ppm (parts per million; a unit that is independent of the main magnetic field) less than that of water protons. At 3T, the Larmor frequency is $f_0 = 127.7$ MHz, resulting in a gap between water and the intrahepatic lipids: 127.7 MHz × $3.25$ ppm or 415 Hz (dephasing). For purposes of comparison, the gap is 208 Hz at 1.5 T.

In gradient echo sequences, there is no rephasing pulse. Thus, when a radiofrequency wave is applied (or magnetic field $B_1$), the water and lipid spins are switched to the transversal plane (if the flip angle $= 90^\circ$). They are then in phase; then during relaxation, there will be progressive dephasing between the spins of the water and lipid protons because of their Larmor frequency of 415 Hz at 3T (208 Hz at 1.5 T), until the signal is received at each echo time.

In the transverse plane, the two vectors progressively dephase with time, i.e., every time that their angle increases (fig. 3). This gap is actually the phase gap, observed in the transverse plane of magnetization. At the end of each cycle, the two vectors of water and lipids are in phase, and at each half cycle they are in opposed phase.

The echo times (TEs) correspond to data using the formula:

$$TE_p = \frac{n}{2} \cdot CS_{water-lipids} \cdot f_0$$

where $CS_{water-lipids}$ represents the frequency gap water and lipids ($CS_{water-lipids} = 3.25 \times 10^{-6}$), which gives the first IP (in-phase) echo at $TE_1 = 1.20$ ms (OP), $TE_2 = 2.40$ ms (IP), $TE_3 = 3.60$ ms (OP), etc. At 1.5 T, the echo times are double: $TE_1 = 2.40$ ms (OP), $TE_2 = 4.80$ ms (IP), $TE_3 = 7$ ms (OP), etc.

However, these theoretical calculations take into account the intensity of the main magnetic field $B_0$, which is always a bit different from what is announced by the manufacturer. This is why $TE_1 = 1.23$ ms (OP) (and not $TE_1 = 1.20$ ms) is recommended by Siemens on our 3T Siemens TRIO TIM imager. In addition, this can provide an indirect calculation of the field $B_0$.

The signal received is always positive and, when the water and lipid protons are in phase ($TE_{OP} = 2.46$ ms, 4.92 ms, 7.38 ms… to 3T), corresponds to the sum of their signal. On the other hand, when the water and lipid protons are in opposed phase ($TE_{OP} = 1.23$ ms, 3.69 ms, 6.15 ms, etc. at 3T), the signal received is equal to the difference of their signal, since the signal intensity reflects the magnitude of the vector and not its phase (25).

As a consequence, the proportion of lipid is equal to the difference of the IP signal minus the OP signal divided by the IP signal, the result divided by 2, as illustrated in figure 4.

**The T2* effect: not to be forgotten**

The classic assertion that “the signal loss in opposed-phase demonstrates the presence of lipids” and its reciprocal “if there are lipids in the liver there is a signal loss on the OP sequences compared to the IP sequence” can easily be confusing because they ignore an inescapable phenomenon in NMRI: T2* relaxation.

Indeed, the signal on the IP and OP images is measured in two different times, i.e., between these two TEs, the transverse magnetization of the water and lipid protons always decreases along a decreasing exponential curve corresponding to the T2* curve of the tissue. The T2* of the normal liver...
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and fatty material is approximately 15 ms and 8 ms, respectively at 3T (unpublished data); consequently, the signal of each drops according to the formula (fig. 5):

\[ S(TE) = k \cdot \exp\left(-\frac{TE}{T_{2}^{*}}\right) \]  

Therefore, if the \( T_{2}^{*} \) is greater than \( T_{E,OP} \), the OP loss due to water and lipid proton dephasing (principle of the IP/OP sequence) is partially or totally compensated by the IP loss because of \( T_{2}^{*} \) relaxation. With moderate steatosis or if the \( T_{2}^{*} \) effect is ignored, one can erroneously conclude that there is no steatosis. This is even more significant at 3T, since the \( T_{2}^{*} \) of a substance is shorter at 3T than at 1.5T, which increases the signal loss caused by \( T_{2}^{*} \) relaxation.

For example, at 3T a loss of \([S(1.23) - S(2.46)]/S(1.23) = 7.9\%\) (formula 3 with \( T_{2}^{*} = 15 \text{ ms} \)) of the signal between the OP (\( T_{E,OP} = 1.23 \text{ ms} \)) and IP (\( T_{E,IP} = 2.46 \text{ ms} \)) sequences is observed, for a liver with \( T_{2}^{*} = 15 \text{ ms} \).

If the \( T_{2}^{*} \) of the liver is shortened, as during hepatosiderosis observed in cirrhosis or even more in cases of hemochromatosis, the signal loss is \([S(1.23) - S(2.46)]/S(1.23) = 18.5\% \) (Equation 3 with \( T_{2}^{*} = 6 \text{ ms} \)) for a hemochromatosis liver with \( T_{2}^{*} = 6 \text{ ms} \). In these cases, if \( T_{2}^{*} \) is not taken into account, an absence of steatosis will be concluded, even if the steatosis is actually quite substantial.

On the contrary, if a \( T_{E,IP} \) less than \( T_{E,OP} \) is chosen, even in total absence of steatosis, a signal loss in opposed-phase will be observed (because of the \( T_{2}^{*} \) relaxation) and the erroneous conclusion of steatosis is made by ignoring the \( T_{2}^{*} \) effect.

Thus, if \( T_{2}^{*} \) relaxation is integrated into the dephasing of lipid and healthy liver protons (water protons), the signal obtained corresponds to a sinusoid damped by a decreasing exponential curve (fig. 6). It is proportional to:

\[
\%{\text{fat}} \cdot \exp\left(-\frac{TE}{T_{2}^{*}\text{fat}}\right) \cdot \cos\left(TE \cdot CS_{\text{water-fat}} \cdot 2\pi\right) \\
+ \%{\text{healthy liver}} \cdot \exp\left(-\frac{TE}{T_{2}^{*}\text{healthy liver}}\right)
\]

The minimums (OPN) and maximums (IPN) correspond to the opposed-phase and in-phase echo times (OP1 = 1.23 ms, IP1 = 2.46 ms, OP2 = 3.69 ms, IP2 = 4.92 ms on our 3T imager, etc.)

Optimization of the TEs at 3T

At 1.5T, most machines use the first two TEs (\( T_{E,OP} = 2.4 \text{ ms} \), \( T_{E,IP} = 4.8 \text{ ms} \) on our...
old 1.5 T imager) with an TE_{OP} < TE_{IP}, with no technical problems. At 3T, however, the first two TEs (TE_{OP} = 1.23 ms, TE_{IP} = 2.46 ms) require substantially reducing the spatial resolution and using very high passbands (PBs), prohibitive since the noise-to-signal ratio is then greatly reduced (noise/signal proportional to $\frac{1}{\sqrt{BP}}$). Some recommend using the first TE_{OP} (1.23 ms) and the second TE_{IP} (4.92 ms) (25), which retains the classic order of the TEs (TE_{OP} < TE_{IP}), but at the cost of a very large T2* effect since the two TEs are distant. With these parameters and if the liver’s T2* is 15 ms (which corresponds to a nonsteatotic liver), there is a 21.8% loss (only by T2* relaxation and not by out-phase) between IP and OP, which clearly complicates image interpretation when searching for steatosis. On our Siemens Trio TIM, we chose to use a 2.46-ms TE_{IP} and a 3.69-ms TE_{OP} so as to use a low passband and good spatial resolution (320 × 208), but which minimizes the T2* effect (7.9% of relative loss with the same T2*), even if the TE order is reversed.

**Imaging**

The theoretical data discussed above should provide a better comprehension of IP/OP imaging and prevent errors in interpretation.

The in-phase/opposed-phase is a dual gradient-echo sequence. This has evolved from the Dixon sequence so that IP and OP images could be compared more precisely since they are acquired during the same flip gradient, at exactly the same slice level.

**Practical considerations**

To easily recognize an OP sequence, one can look for the India ink artifact (26), i.e., the parenchyma surrounded by a black line (hyposignal), when the parenchyma are encircled by fat (fig. 7). This artifact is not a chemical shift artifact, which corresponds to a spatial coding error in the frequency coding direction (only) because of the difference in precession frequency between the water and fat protons. In this case, the black line on the organ borders is only observed in the frequency coding direction, and only on one side, since this is actually a gap (fig. 7). On the other side there is a white line on the border. It should be noted that this chemical shift artifact is increased at 3T compared to 1.5T, and is increased if the passband is increased (but to the detriment of the signal-to-noise ratio).

The India ink artifact represents voxels containing 50% lipids and 50% water, at the interfaces between different tissues (liver/mesenteric fat, kidney/retroperitoneal fat, etc.). This India ink artifact is decreased when the spatial resolution is increased.

In-phase/opposed-phase image interpretation should take several data into account:

- What is the order of the TE_{IP}s and TE_{OP}s?

A signal loss can be explained on the sequence with the longest TE, but only based on the T2* effect.

- Can the liver’s or the investigated lesion’s T2* be very short?

In this case, T2* relaxation should not be considered as insignificant and can, following the order of the TEs, suggest steatosis or, on the other hand, mask true steatosis.

- Is the windowing of each sequence the same?

If not, comparisons with the naked eye are difficult to make.

To avoid the windowing problem, several authors (27, 28) have reported using subtraction phase – opposed-phase images, which quickly demonstrates focal lesions (fig. 7) or identifies steatosis. This can be shown by comparing the liver signal to the spleen signal (when there is no spleen steatosis and not forgetting the T2* effect), or to the muscle signal.

![Fig. 7:](https://example.com/f7.png)

**IP sequence (arrow = chemical shift artifact: hyposignal band bordering the spleen on one side)**

**OP sequence (arrowhead = India ink artifact with recognition of the opposed-phase; arrow = signal loss of a liver adenoma)**

**IP – OP subtraction image. The adenoma is clearly visible**
Detection and characterization of focal lesions

Two types of lesions are often distinguished: low (e.g., focal steatosis) and high (e.g., lipoma) lipid load. Fat saturation (Fat-sat) is classically used to screen and characterize high lipid loads, because in this case, the proportion of fat is very high and therefore gives a strong signal loss in Fat-sat. On the other hand, in IP/OP there is a slight signal loss (independently of T2* relaxation) because of the very low proportion of the water proton signal compared to the fat protons, predominant in this type of lesion. However, there is definitely a signal loss (one can measure the signal of the retroperitoneal or subcutaneous fat on the IP/OP sequence) because the adipocytes necessarily contain a certain proportion of water. In contrast, in lesions with a low fat load, the IP/OP sequence appears more sensitive than saturation of the fat signal because the signal loss is, theoretically, double that obtained in Fat-sat (29), since \(|\text{Signal}_{\text{water}} + \text{Signal}_{\text{lipid}}|\) and \(\text{Signal}_{\text{water}} - \text{Signal}_{\text{lipid}}|\) are being compared (fig. 4). It should be remembered, however, that signal loss does exist in Fat-sat, including for these low lipid loads.

Thus, the IP/OP sequence can detect focal lesions or pseudolesions containing lipids (fig. 7) or provide arguments for characterization (27). Several lesions frequently contain lipids (30):

- Benign lesions: lipoma, xanthomatous lesions of Langerhans cell histiocytosis, epiploplasty, adenoma, hepatic adrenal rest tumor, angiomyolipoma, teratoma, focal steatosis, and pseudotumoral steatosis.
- Malignant lesions: primary liposarcoma, metastasis of liposarcoma, hepato-cellular carcinoma, metastases (very rare).

In addition, detection of steatosis may have a prognostic role in the future. Recently, two French studies (31, 32) have investigated the genetic mutations present in liver adenomas. One of them studied the HNF1α gene, accounting for highly steatotic adenomas in which the risk of degeneration to cancer is low (approximately 7%), thus giving the IP/OP sequence both a diagnostic and prognostic potential for these rare benign tumors.

Quantification

Before quantifying hepatic steatosis, two problems must be solved: the ambiguity related to lipid overloads greater than 50% and the T1 parasite effect of quantification.

### Ambiguity

In opposed-phase images (and independently of the T2* effect), the signal is collected in magnitude, i.e., in the absolute value of the water proton signal minus the lipid proton signal. Hence, a 33% lipid load, for example, gives the same signal as a 67% load, thus creating an ambiguity in the interpretation of the steatotic load (3, 25, 33) (fig. 8). This ambiguity is problematic in imaging because a low lipid load will have the same repercussion on the IP/OP images (and even subtraction images).

### Table I

<table>
<thead>
<tr>
<th>Angle</th>
<th>SIP (ppm)</th>
<th>SOP (ppm)</th>
<th>S_{IPP} - S_{SOP}</th>
<th>Loss to (T2^*) (%)</th>
<th>Steatosis rate (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>20°</td>
<td>290</td>
<td>160</td>
<td>44.8</td>
<td>9</td>
<td>17.9</td>
</tr>
<tr>
<td>60°</td>
<td>318</td>
<td>149</td>
<td>53.1</td>
<td>9</td>
<td>22.1</td>
</tr>
</tbody>
</table>

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as a high load (25). This is even more challenging when one is seeking to quantify the steatosis. An original solution has been suggested (3) and uses what can be called the “T1 parasite effect” of the IP/OP sequence to resolve this ambiguity.

The T1 parasite effect

The T1 sequence of the fat and liver is very different, with approximately 380 ms and 810 ms, respectively, at 3T and 340 ms and 590 ms, respectively, at 1.5T (34). In the dual gradient-echo IP/OP, flip angles around 60° at 3T (Ernst angle and 70° at 1.5 T) are often used, so that there is a maximum signal and therefore a good noise-to-signal ratio. However, with a 60° angle at 3T (70° at 1.5T), T1 weighting is not insignificant (the more the flip angle α approaches 90°, the higher the T1 weighting is). The water and lipid photon magnetization vectors also push depending on the longitudinal axis (of βo) between each echo time (TEIP and TEOP), at a different speed for each constituent since each one possesses a different T1. Finally, signal collection during TEIP and TEOP takes into account the presence or absence of lipids (an opposed-phase phenomenon), T2* relaxation, and possibly the T1 parasite effect.

The latter must therefore be taken into account to precisely quantify hepatic steatosis or one can attempt to minimize it using a 20° flip angle, as proposed by Hussain et al. (3). This low flip angle can remove the weighting of the T1 sequence. The gradient-echo signal is given by:

\[ S = K \left( \frac{1 - \exp(-TR/T1)}{\sin\theta \cdot \exp(-TE/T2*)} - \frac{1 - \exp(-TR/T1)}{\cos\theta} \right) \]

The in-phase/out-phase signal is given by:

\[ S_{IP} = (1 - P) \cdot S_{\text{healthy liver}} + P \cdot S_{\text{fat}} \]
\[ S_{OP} = (1 - P) \cdot S_{\text{healthy liver}} + P \cdot S_{\text{fat}} \]

(with P = percentage of true steatosis) and the percentage of steatosis calculated by:

\[ \% \text{calculated steatosis} = \frac{S_{IP} - S_{OP}}{2 \cdot S_{IP}} \]

The percentage of calculated steatosis is obtained in relation to the percentage of real steatosis, by replacing in the equations above the parameters T1, T2*, TR with their values for the healthy liver and for fat. Figure 9 shows the curves thus obtained with 20° and 60° angles.

The percentages of steatosis calculated for extreme rates (low or very high) are negative, because there is no correction due to T2* with this method. In particular, it can be observed that the curve obtained with a 60° angle is not exactly correlated between the calculated and true rate of steatosis, with, for example, a 47% calculated steatosis, whereas the true steatosis is 40% (fig. 9). This is caused essentially by the T1 differences in the two constituents.

According to these curves, it is therefore wiser to calculate the percentage of steatosis based on a sequences with a 20° flip angle, i.e., unweighted in T1, thus alleviating the T1 parasite effect. However, at 20° the signal is much weaker than at 60° (for the same TR) and the noise-to-signal ratio is less favorable, which can create a new error. But, as has recently been reported (3), comparing the relative signal loss with a 60° and then a 20° flip angle solves the ambiguity stemming from the lipid loads over or under 50%. When the steatosis rate calculated at 60° is greater than the rated calculated at 20°, we are in the first part of the curve and it can be deduced that the true steatosis rate is less than 50%. On the other hand, if the steatosis rate calculated at 60° is lower than the rate calculated at 20°, there are more lipids than water and the percentage is greater than 50%. The T1 parasite effect therefore becomes an advantage. Finally, quantifying hepatic steatosis requires:

- calculating the patient’s liver’s T2*;
- a dual gradient-echo IP/OP with a 60° flip angle;
- a dual gradient-echo IP/OP sequence with a 20° flip angle.

Practical example

Figures 10 and 11 show the example of a 35-year-old female patient explored with MRI for focal nodular hyperplasia. Comparing IP and OP images did not provide a convincing argument in favor of steatosis. Nonetheless, the IP – OP subtraction image showed a hypersignal of the liver compared to the spleen and the muscles, probably demonstrating hepatic steatosis (the T2* image should be taken into account, as should the TEIP < TEOP). This apparent discordance results from a different visualization window, giving the illusion that the liver’s signal did not drop in OP.

This patient’s T2* liver image was evaluated at 13 ms (measured on a multiecho gradient-echo sequence). Table I gives the result of the signal measurements for each sequence.

Since the percentage of steatosis calculated was higher with a 60° angle, the ambiguity is resolved: the steatosis rate is indeed 17.9% (in the opposite case, it would have been 100 – 17.9 = 82.1%).

A single-voxel magnetic resonance spectroscopy of the proton (PRESS sequence, free breathing, 64 acquisition, 2048 points, without water suppression, TE = 30 ms, TR = 3000 ms) evaluates the ratio of the water peak surface minus the lipid peak surface (CH2) over the water peak surface at 20% (fig. 11) (MRUI software).

The percentage obtained with the IP/OP sequence is therefore quite close to that given by spectroscopy, the current reference method for noninvasive quantitatively evaluation of fatty liver.

Conclusion

The dual gradient-echo in-phase/opposed-phase sequence is a robust and reliable sequence for imaging and quantifying steatosis, on condition that the order of the TEIP and TEOP, T2* relaxation, and possibly the T1 parasite effect are taken into account, notably to resolve the ambiguity beyond 50% of steatosis. The arrival of 3T imagers requires that this sequence be optimized and furthermore requires at 1.5 T that the technical aspects be well in hand to prevent interpretation errors.
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Fig. 10:

a IP sequence at 60°.

b OP sequence at 60°: there is no clear signal loss between the two sequences, leading to a mistaken conclusion that there is no steatosis. This is caused by the different visualization windows, as shown by the spleen signal.

c IP–OP subtraction: the liver is more in hyperintensity than the spleen or the muscles, demonstrating very probable steatosis.

Fig. 11:

The same patient as Fig. 10.

a IP sequence at 20°.

b OP sequence at 20° (used to calculate steatosis by avoiding the T1 parasite effect)

c Single-voxel MRS, evaluating the lipid rate (CH2 peak) in the 20% voxel. The arrows indicate the CH and CH3 peaks of these same lipids.
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